Letter to the Editor

Late onset branch retinal artery occlusion following coronary angiography

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Sir,

There are several cases in the literature describing the different variations of post-embolization syndrome. The skin, the renal and gastrointestinal systems and the retina are the major targets of cholesterol crystals after cardiac catheterization. Greater use of coronary angiography has resulted in a relatively increased risk of these post-cardiac embolizations (Bitti 2000). To our knowledge only a few patients with branch artery occlusion (BRAO) or cilioretina artery occlusion during coronary angioplasty have been reported (Bucci et al. 1989; Dickens et al. 1994; Filatov et al. 1995; Loewenstein et al. 1999). In this case report, we describe a patient with visual loss and branch retinal artery occlusion 24 hours after cardiac catheterization.

A 57-year-old woman was referred for acute onset of blurred vision in her left eye during the morning of the day following her coronary angiography (24 hours after catheterization). She had a history of arterial hypertension, hypercholesterolaemia and diabetes. An ophthalmological examination revealed a visual acuity (VA) of 20/100 with a paracentral scotoma in the left eye; VA in her right eye had been low (20/200) since childhood due to strabismic amblyopia. Anterior segment examination was normal. Fundoscopy examination in the left eye revealed a white embolus (Hollenhorst plaque) partially occluding the superior temporal branch of the central retinal artery, associated with a moderate retinal oedema (Fig. 1), while the right eye was normal. Fluorescein angiography confirmed the non-filling of the superotemporal branch retinal artery (Fig. 2), while visual field testing showed a paracentral visual defect in accordance with the artery occlusion. Twelve months later, VA had improved to 20/32 with a tiny paracentral scotoma without any intervention.

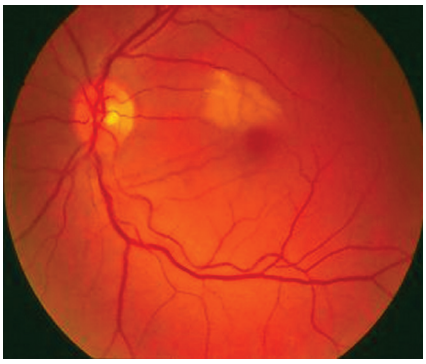

Fig. 1. Fundus photograph of the left eye demonstrates the cholesterol embolism at the superior temporal branch of the central retinal artery, associated with a moderate retinal oedema in the distribution of the vessels.

The development of retinal cholesterol emboli is due to ulceration of an atheromatous plaque, frequently localized in the origin of the aorta; as a result of rupture, plaque contents are released into the arterial circulation. There are two possible mechanisms for late onset of BRAO following coronary angioplasty. First, there is a possibility of the late release (second phase) of systemic cholesterol emboli after angioplasty that can cause a delayed BRAO. A second mechanism may involve the partial artery obstruction caused by atherosclerotic plaque (cholesterol emboli) at the time of coronary artery catheterization that becomes symptomatic with visual loss after complete artery occlusion due to the delayed inflammatory response. In this case, VA recovered a few months later without any intervention, leaving only a small paracentral scotoma.

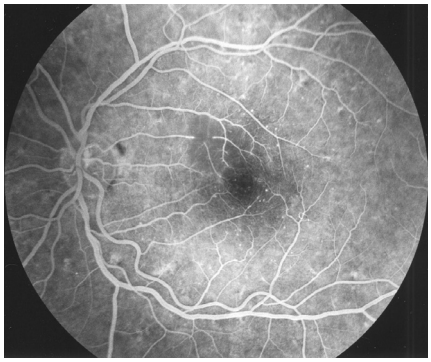

Fig. 2. Fluorescein angiogram of the left eye shows the non-filling of the superotemporal branch retinal artery.

With the increasing incidence of invasive cardiac procedures, cardiologists and ophthalmologists should increase their awareness of this post-cardiac catheterization complication. Visual symptoms and retinal artery occlusions caused by embolization in patients after coronary angiography could appear 24 hours after catheterization.

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